EJMI

DOI: 10.14744/ejmi.263065
EJMI 2026;10(1):65–72

Research Article

Trimester-Specific and Dose-Dependent Associations Between Maternal Smoking and Neonatal Birth Weight: A Population-Based Analysis of the CDC WONDER Natality Database (2016–2024)

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Abstract

Objectives: Maternal smoking during pregnancy impairs fetal growth, yet the relative contribution of trimester-specific exposure and cessation timing remains unclear. Quantitative trimester-based analyses that integrate cigarette dose and neonatal outcomes are limited.

Methods: This retrospective, population-based study used data from the CDC WONDER Natality database (United States, 2016–2024). Women who reported smoking at the beginning of pregnancy and had complete trimester-specific data were included. Smoking exposure was analyzed both categorically and quantitatively (cigarettes per day). The primary outcome was neonatal birth weight (grams); a secondary analysis assessed small-for-gestational-age (SGA) among term deliveries. Multivariable linear regression was adjusted for maternal age, pre-pregnancy BMI, gestational weight gain, interpregnancy interval, and pregnancy-related morbidities. ROC analysis identified discriminatory thresholds.

Results: Among 4,931 pregnancies, second-trimester smoking showed the strongest independent negative association with birth weight (B=−1.246 g per cigarette; p<0.001), followed by first-trimester smoking (B=−0.957 g; p<0.001). Third-trimester smoking had minimal clinical impact. Second-trimester cessation was associated with higher birth weight (p<0.001), whereas third-trimester cessation showed no benefit. Among term births, only first-trimester smoking was associated with SGA (p=0.022). A threshold of 15 cigarettes per day predicted SGA with a sensitivity of 71% and a specificity of 75%.

Conclusion: Smoking timing critically determines fetal growth impact, underscoring the importance of very early cessation.

Keywords: Birth weight, Maternal smoking, Smoking cessation

Cite This Article: *Mutlu Sütçüoğlu B, Karçaaltuncaba C, Karçaaltuncaba D. Trimester-Specific and Dose-Dependent Associations Between Maternal Smoking and Neonatal Birth Weight: A Population-Based Analysis of the CDC WONDER Natality Database (2016–2024). EJMI 2026;10(1):65–72.*

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Submitted Date: January 24, 2026 **Revision Date:** March 13, 2026 **Accepted Date:** March 16, 2026

Optimal fetal growth depends on a complex interplay of physiological and pathological determinants. Among the physiological factors, maternal comorbidities—particularly pre-gestational body mass index (BMI) and smoking status—have been directly associated with birth weight.[1]

Maternal tobacco use remains one of the most preventable yet persistent causes of adverse pregnancy outcomes worldwide. Despite a global decline in smoking prevalence, up to 10–15% of pregnant women continue to smoke during pregnancy, with considerable regional variation and differing cessation patterns across trimesters.[2] The detrimental effects of in utero tobacco exposure on fetal growth and birth outcomes have been extensively documented, including an increased risk of low birth weight, intrauterine growth restriction (IUGR), and small-for-gestational-age (SGA) infants. However, growing evidence indicates that both the timing and amount of cigarette exposure during gestation play a critical role in determining the extent of fetal growth impairment. Although the mechanisms by which maternal smoking adversely affects pregnancy outcomes are not fully elucidated, proposed pathways include reductions in placental perfusion and alterations in fetal epigenetic regulation with abnormal methylation.[3]

Meta-analyses and cohort studies suggest that fetal growth restriction becomes evident after the first trimester, particularly affecting head circumference and femur length in the second trimester and estimated fetal weight in the third.[4] Bailey et al.[5] demonstrated a dose-dependent relationship, where smoking more than ten cigarettes per day was assciated with a more than 10-percentile reduction in estimated fetal weight and head circumference during mid-gestation. Similarly, Blatt et al.[6] reported that continued smoking through the second and third trimesters more than doubled the risk of fetal growth restriction, while cessation during the first trimester substantially mitigated this risk. Longitudinal analyses from the Millennium Cohort Study confirmed that quitting by the end of the first trimester normalizes birth weight to levels comparable with nonsmokers, whereas cessation later in pregnancy only partially reverses growth deficits.[7] In contrast, ongoing smoking into late pregnancy exerts cumulative adverse effects on fetal growth, with the highest risk for SGA observed after 33 weeks of gestation. These findings emphasize that both smoking intensity and the gestational timing of cessation are critical determinants of neonatal outcomes. However, limited research has integrated trimester-specific smoking patterns and quantitative measures of cigarette exposure with direct comparisons to neonatal birth weight.[8]

Accordingly, the primary aim of the present study was to enhance the explanatory capacity of statistical models by focusing specifically on women who reported smoking at the beginning of pregnancy, rather than conducting a conventional comparison between smokers and non-smokers. A second aim was to address a persisting gap in the literature: although smoking cessation during pregnancy is consistently recommended—because smoking is associated with morbidities and cessation is cost-effective—there remains no consensus regarding the gestational periods when smoking exposure most significantly influences fetal growth. This study is based on data retrieved from the Centers for Disease Control and Prevention (CDC) WONDER natality database, a large, population-based registry that captures detailed birth certificate information across the United States. The database uniquely provides trimester-specific, quantitative measures of maternal cigarette consumption alongside comprehensive neonatal birth data, allowing for robust evaluation of timing- and dose-dependent effects of smoking on fetal growth.

Methods

This retrospective, population-based study used data from the Centers for Disease Control and Prevention (CDC) Wide-ranging Online Data for Epidemiologic Research (WONDER) natality database. CDC WONDER is a publicly accessible, de-identified national surveillance platform that provides comprehensive birth certificate data derived from the National Vital Statistics System (NVSS). We used the "Births" expanded natality files for the years 2016 through 2024, which include detailed maternal, pregnancy, and neonatal variables. All recorded live births occurring in the United States between January 1, 2016, and December 31, 2024, were eligible for inclusion. Because the dataset is publicly available and contains no personal identifiers, this study was exempt from institutional review board approval and informed consent requirements.

All recorded live births occurring in the United States between January 1, 2016 and December 31, 2024 were eligible for inclusion. No a priori restrictions were applied based on maternal race/ethnicity, geographic region, mode of delivery, congenital anomalies, maternal comorbidities, infections, educational status, paternal characteristics, use of assisted reproductive technology (ART), prior obstetric history, labor complications, or infant characteristics. The natality public-use data files were downloaded and analyzed using statistical software rather than the CDC WONDER query interface. Therefore, no automated cell suppression was applied. Records with missing data for primary exposure or outcome variables were excluded from regression analyses using a complete-case approach. The primary exposure of interest was maternal cigarette smoking during pregnancy, categorized by trimester. CDC WONDER pro-

vides trimester-specific self-reported smoking data, including the number of cigarettes smoked per day during the first, second, and third trimesters.

Smoking exposure was evaluated in two complementary ways:

1. Binary exposure status(yes/no)within each trimester.
2. Quantitative exposure, defined as the average number of cigarettes smoked per day in each trimester.

In addition to trimester-specific smoking status, cessation patterns during pregnancy were categorized into three groups: women who discontinued smoking in the first, second, or third trimester.

The following maternal characteristics were collected: pre-pregnancy BMI, total gestational weight gain, interpregnancy interval, maternal age, and pregnancy-related complications, including gestational diabetes or hypertension, eclampsia, and preeclampsia. Gestational weight gain was analyzed as a categorical variable and stratified into five predefined groups (<5 kg, 5–9 kg, 10–14 kg, 15–18 kg, and ≥19 kg) to account for potential non-linear associations with birth weight outcomes. The primary outcome of the study was neonatal birth weight (grams), analyzed as a continuous variable for all recorded live births during the study period. As a secondary analysis, the cohort was restricted to term pregnancies (≥37 weeks of gestation). Within this subgroup, neonates were categorized into two groups based on birth weight: low birth weight (<2500 g) and non–low birth weight (≥2500 g). In this study, small for gestational age (SGA) among term births (≥37 weeks) was defined as a birth weight <2500 g, as calculating gestational age–specific birth-weight percentiles for each observation was not feasible in this large population-based dataset.

This study used the Centers for Disease Control and Prevention (CDC) WONDER natality database, a publicly accessible de-identified dataset. Since the data are anonymous and publicly available, the study was exempt from institutional ethics committee approval and informed consent requirements in accordance with international research regulations.

Statistical Analysis

All statistical analyses were performed using SPSS Statistics version 21.0 (Armonk, NY: IBM Corp., USA). Descriptive statistics were used to summarize maternal, pregnancy, and neonatal characteristics. Continuous variables were reported as mean ± standard deviation (SD) or median with range, depending on distribution. Categorical variables were presented as frequencies and percentages. Comparisons between two independent groups were performed using the Mann–Whitney U test. For comparisons involving more than two independent groups, the Kruskal–Wallis test was used. When applicable, post hoc pairwise comparisons were conducted with appropriate adjustment for multiple testing. Correlations between continuous variables were assessed using Spearman's rank correlation coefficient. Receiver operating characteristic (ROC) curve analysis was performed to determine optimal cut-off values for relevant continuous variables. The area under the curve (AUC), 95% confidence intervals (CIs), and Youden's index were calculated to identify the most discriminative thresholds. For analyses of the primary outcome, multivariable linear regression models were constructed to evaluate the independent association between trimester-specific smoking exposure and neonatal birth weight. Regression coefficients(B),95% CIs, and p-values were reported. A p-value <0.05 was considered statistically significant.

Results

Study Population

A total of 4,931 pregnant women who reported smoking at the beginning of pregnancy and had complete smoking data for all three trimesters were included in the analysis. The mean maternal age was 28 years (SD±1), the median gestational age at delivery was 38 weeks (range: 18 weeks), and the median pre-pregnancy BMI was 27.21 kg/m^2 (range: 22.34). The median neonatal birth weight was 3,044 g (range: 3,220 g). The median interval since the last pregnancy was 55.2 months (range: up to 137 months). A total of 270 women (5.2%) experienced at least one pregnancy-related morbidity, while 4,895 women (93.6%) experienced no morbidities. A total of 845 women (17.1%) gained ≤5 kg during pregnancy, while 919 women (18.6%) gained 5–9 kg. Moderate weight gain of 10–14 kg was observed in 1,053 women (21.4%). Additionally, 973 women (19.7%) gained 15–18 kg, and the highest weight-gain category (19–44 kg) included 1,142 women (23.2%). This distribution indicates that nearly one-quarter of the cohort experienced excessive gestational weight gain, whereas approximately one in five experienced insufficient gestational weight gain. The median number of cigarettes smoked per day was 10 (range: 98) in the first trimester, 6 (range: 98) in the second trimester, and 4 (range: 98) in the third trimester, respectively.

Correlation Analyses

Neonatal birth weight was significantly negatively correlated with maternal age ($r=-0.314$, $p<0.001$) and with pre-pregnancy BMI ($r=-0.392$, $p<0.001$). In contrast, gestational weight gain demonstrated a strong positive correla-

tion with birth weight (r=0.672, p<0.001). No association was observed between interpregnancy interval and birth weight (p=0.255). Birth weight was significantly lower in infants born to women with at least one morbidity compared with those without morbidity (median 2,981 g vs. 3,053 g; p<0.001).

Smoking Cessation and Birth Weight

A total of 431 (8.7%) pregnant women who quit smoking during the second trimester. In the second trimester, birth weight differed significantly across smoking categories: those who quit smoking had higher birth weights than persistent smokers (median 3,144 g vs. 3,033 g; p<0.001).

A total of 612 pregnant women (12.4%) quit smoking in the third trimester. In the third trimester, there was no significant difference in birth weight between women who quit smoking and those who continued smoking (p = 0.644). Median birth weights were 3,045 g for women who quit smoking and 3,033 g for women who smoked persistently.

Multivariable Linear Regression Analysis

A multivariable linear regression model, adjusted for maternal age, pre-pregnancy BMI, gestational weight gain, morbidities, and trimester-specific smoking, explained 48.1% of the variance in birth weight (R^2=0.481) (Table 1). The Durbin–Watson statistic indicated no autocorrelation in model residuals. Gestational weight gain was the strongest positive predictor of birth weight. Each 1-kg increase was associated with an average 109-g gain (B=109.1 g; 95% CI 104.9–113.3; p<0.001). Pre-pregnancy BMI was also a significant positive predictor (B=21.0 g per unit; 95% CI 19.0–23.1; p<0.001). Conversely, pregnancy morbidity (B=−69.2 g; 95% CI −86.3 to −52.2; p<0.001) and maternal age (B=−10.7 g per year; 95% CI −13.7 to −7.6; p<0.001) were associated with decreased birth weight.

The effect of trimester-specific smoking remained significant after adjustment. Second-trimester smoking showed an independent negative association with birth weight (B=−1.246 g per cigarette; 95% CI −1.82 to −0.68; p<0.001). First-trimester smoking had a weaker but still significant negative effect (B=−0.957 g; 95% CI −1.35 to −0.56; p<0.001). Third-trimester smoking was associated with a small positive coefficient (B=0.626 g; 95% CI 0.14–1.11; p=0.012), which is clinically negligible and likely reflects residual confounding or a statistical artifact.

SGA Analysis in Term Deliveries

Among women who delivered after 37 weeks of gestation, infants were stratified by SGA status using a birthweight threshold of <2500 g (Table 2). In this subgroup, only first-trimester smoking was significantly associated with SGA status (p=0.022). The median number of cigarettes smoked during the first trimester was 20 (range: 37) and 9 (range: 98) in the SGA and non-SGA groups, respectively. No significant associations were observed for second-trimester smoking (median 10 vs. 6; p=0.340) or third-trimester smoking (median 10 vs. 4; p=0.153). ROC analysis assessing the relationship between first-trimester cigarette consumption and SGA demonstrated significant discriminative ability: a threshold of 15 cigarettes yielded 71% sen-

Table 2. Trimester-specific smoking and SGA status

	Birth weight (gr)		p
	<2500	>2500	
	Median (range)	Median (range)	
First trimester	20 (37)	9 (98)	0.022
Second trimester	10 (39)	6 (98)	0.340
Third trimester	10 (39)	4 (98)	0.153

SGA: Small-for-gestational-age.

Table 1. Multivariable linear regression predicting birth weight

Variable	B (95% CI)	SE	β	t	p	Zero	Partial	Part	VIF
(Constant)	2448.795 (2341.437-2556.154)	54.761		44.718	**<0.001**				
Maternal weight gain	109.089 (104.895-113.282)	2.139	0.874	50.994	**<0.001**	0.604	0.546	0.391	2.558
Pre-pregnancy BMI	21.036 (19.032-23.041)	1.022	0.347	20.575	**<0.001**	0.293	0.22	0.404	2.475
Pregnancy morbidity	-69.207 (-86.265- -52.150)	8.700	-0.086	-7.955	**<0.001**	-0.117	-0.085	0.974	1.026
Maternal age	-10.668 (-13.695 - -7.641)	1.544	-0.081	-6.908	**<0.001**	-0.102	-0.074	0.843	1.186
First trimester smoking	-0.957 (-1.354 - -0.561)	0.202	-0.073	-4.735	**<0.001**	-0.07	-0.051	0.488	2.047
Second trimester smoking	-1.246 (-1.817 - -0.675)	0.291	-0.079	-4.276	**<0.001**	-0.063	-0.046	0.337	2.967
Third trimester smoking	0.626 (0.139-1.113)	0.249	0.037	2.518	**0.012**	0.037	0.027	0.54	1.851

B: Unstandardized regression coefficient; CI: Confidence interval; SE: Standard error; β: Standardized regression coefficient; VIF: Variance inflation factor; BMI: Body mass index.

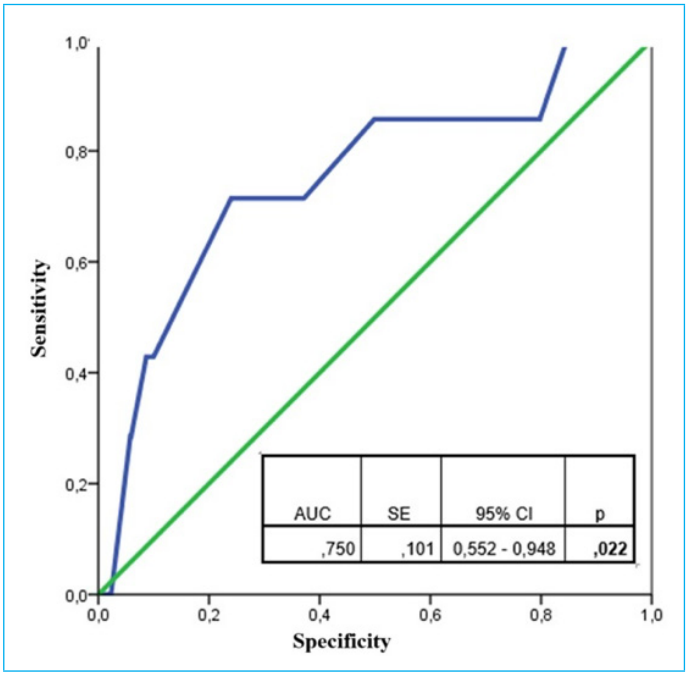

Figure 1. Receiver operating characteristic (ROC) curve for first-trimester cigarette consumption predicting small-for-gestational-age (SGA) among term deliveries.

The ROC curve demonstrates the discriminative ability of first-trimester cigarette consumption to predict SGA status. The area under the curve (AUC) was 0.750 (SE 0.101; 95% CI 0.522-0.948; p=0.022), indicating moderate predictive performance. A threshold of 15 cigarettes per day yielded 71% sensitivity and 75% specificity. The diagonal reference line represents no discriminatory ability (AUC=0.50).

sitivity and 75% specificity (p=0.022) (Fig. 1). No significant discriminatory performance was observed for smoking in the second or third trimesters (p=0.342 and p=0.155, respectively).

Discussion

In this large cohort of pregnant women with trimester-specific smoking data, our findings demonstrate distinct temporal effects of smoking exposure and smoking cessation on fetal growth. The most clinically relevant observation was that smoking cessation during the second trimester was associated with a significant improvement in neonatal birth weight, whereas cessation during the third trimester did not confer a measurable benefit. Women who discontinued smoking during the second trimester delivered infants with significantly higher birth weights than women who continued smoking. In contrast, cessation during the third trimester did not translate into a statistically or clinically meaningful difference in birth weight.

Importantly, our study provides novel evidence regarding the association between trimester-specific smoking exposure and the risk of SGA among term deliveries. To our knowledge, this is the first report demonstrating that in term pregnancies, only smoking in the first trimester was significantly associated with SGA status, whereas smoking in the second and third trimesters was not independently associated with SGA in this subgroup. Given that the first trimester corresponds to the critical period of placentation and the early establishment of uteroplacental circulation, smoking during this developmental window may impair placental vascularization and trophoblastic invasion, thereby increasing the likelihood of fetal growth restriction.

The association between maternal smoking and impaired fetal growth is well established in the literature. Compared with non-smokers, smoking during any trimester has been shown to increase the risk of fetal growth restriction, and infants born to mothers who smoke weigh on average 150–300 g less at birth.[9] Approximately one-quarter of cases of fetal growth restriction are attributable to maternal smoking. Although differences between smokers and non-smokers are consistently demonstrated, trimester-specific exposure patterns and the timing of smoking cessation remain areas of ongoing debate.

Preconceptional smoking has generally been considered to have limited impact on birth weight if cessation occurs early in pregnancy.[6] A large U.S. cohort study demonstrated that smoking cessation during the first trimester reduced SGA rates by 31% among preterm births and by 55% among term births. Although second-trimester cessation was also associated with reductions in SGA in both term and preterm deliveries, its effect was reported to be less statistically robust than cessation in the first trimester.[10] Our findings are largely consistent with these observations. However, in contrast to prior studies, we evaluated birth weight and SGA as separate outcomes using distinct statistical models, thereby providing a more granular assessment of trimester-specific effects. In our cohort, first-trimester smoking increased the probability of SGA, whereas cessation during the second trimester was associated with improved birth weight. Third-trimester cessation, however, did not appear to influence neonatal weight.

From a biological perspective, our trimester-specific findings are consistent with the known timeline of placental development. The first trimester represents the critical window of decidual invasion, spiral artery remodeling, and early establishment of uteroplacental circulation. Disruption during this period may lead to structural and functional alterations in placental vascularization that persist throughout gestation. Several studies suggest that maternal smoking does not necessarily reduce overall placental weight; rather, it induces microvascular and functional remodeling.

[11] Increased resistance in umbilical blood flow, abnormal mechanical properties of chorionic villi, enhanced vasoconstrictive responses to endothelin-1, and reduced capillary surface area have all been described in placentas exposed to tobacco smoke.[12] Such early architectural changes may explain why first-trimester smoking in our cohort was independently associated with SGA among term births, even when smoking later in pregnancy did not show a similar association.

Importantly, early exposure may also induce epigenetic modifications that extend beyond transient vascular effects.[13] Maternal smoking has been associated with differential DNA methylation patterns, including characteristic "smoking-related methylation signatures" in genes involved in growth regulation and placental function. Evidence suggests that part of the effect of maternal smoking on birth weight is mediated through DNA methylation changes.[14] Therefore, smoking during the first trimester may not simply exert a temporary toxic influence but may instead trigger persistent molecular alterations that limit fetal growth potential throughout pregnancy. This mechanism provides a biologically plausible explanation for the stronger association between first-trimester exposure and SGA risk observed in our study.

In contrast, smoking exposure during the second trimester occurs after primary placental vascularization has largely been established. Although continued exposure may still impair nutrient transfer and fetal growth velocity, these effects may be at least partially reversible if smoking cessation occurs before late gestation. This interpretation aligns with our finding that second-trimester cessation was associated with increased birth weight, whereas third-trimester cessation did not confer measurable benefit. Once placental architecture and growth trajectories are established, late behavioral modification may have a limited capacity to restore normal fetal growth.

Our finding that second-trimester smoking cessation positively influences birth weight is supported by previous literature. Several studies have demonstrated that smoking cessation or reduction during the second trimester improves neonatal weight outcomes.[15] In a large UK study analyzing approximately 11,000 births, the gestational age cut-off for achieving birth weights comparable to those of non-smokers was identified as 16 weeks.[16] Smoking cessation beyond this time point did not significantly improve birth weight, suggesting that exposure after early placentation may have only partially reversible effects, and that delayed cessation limits recovery potential. Taken together, our findings and the existing literature suggest that smoking during the second trimester—after placental vascularization has been established—may exert a partially reversible effect on fetal growth, thereby influencing neonatal birth weight.

A key strength of our study is the advanced trimester-specific quantitative analysis of cigarette exposure. Second-trimester smoking demonstrated an independent and more pronounced negative association with birth weight, whereas first-trimester smoking showed a weaker yet statistically significant effect. Each additional cigarette smoked during the second trimester was associated with an approximately 1.25-g reduction in neonatal birth weight, while each cigarette smoked during the first trimester was associated with an approximately 1-g reduction. To our knowledge, this is the first study to provide a trimester-specific, per-cigarette quantitative estimate of the impact on birth weight. Most prior studies have examined correlations between overall cigarette consumption and birth weight without stratifying by trimester. A linear relationship between the number of cigarettes smoked and low birth weight has been consistently demonstrated. For example, in a study of 1,400 pregnancies, infants born to mothers who smoked 6–10 cigarettes per day weighed approximately 320 g less than those of non-smokers, whereas infants of mothers who smoked 11–40 cigarettes per day weighed approximately 435 g less.[17] In another study evaluating 4,211 pregnancies, smoking ≥25 cigarettes per day was associated with a 289 g reduction in birth weight compared with non-smoking.[18] However, these studies did not incorporate trimester-specific exposure data.

Additionally, many studies have focused on urinary cotinine measurements as an objective biomarker of smoking exposure. Although cotinine levels correlate linearly with birth weight reduction, biomarker-based screening is costly and time-consuming. Importantly, prior research has demonstrated a strong correlation between self-reported cigarette consumption and urinary cotinine levels. Therefore, our finding that first-trimester exposure to approximately 15 cigarettes per day significantly increases the likelihood of SGA provides a clinically interpretable and cost-effective metric for patient counseling.

The strengths of our study include a large cohort size with trimester-specific smoking assessment; standardized recording of birth weight, gestational age, and pregnancy complications; and use of a reliable population-based data source. These features allowed us to evaluate trimester-specific effects on birth weight and SGA separately, providing a more nuanced interpretation of fetal growth outcomes. Limitations should also be acknowledged. The cohort reflects a specific ethnic population, which may limit generalizability. Furthermore, smoking exposure was based on self-report, introducing potential reporting bias.

Conclusion

Overall, our findings suggest that the second trimester represents a critical window during which modification of maternal smoking behavior may still positively influence fetal weight gain. However, exposure during the first trimester appears to have a stronger and potentially irreversible impact on SGA risk, likely mediated through early placental and epigenetic mechanisms. These results underscore the importance of very early smoking cessation—ideally before conception or immediately after pregnancy recognition—not only to optimize birth weight but also to reduce the risk of term SGA. The timing of exposure is therefore crucial. Early initiation of prenatal care may play a pivotal role in supporting smoking cessation efforts during this vulnerable developmental period.

Disclosures

Ethics Committee Approval: This study used the Centers for Disease Control and Prevention (CDC) WONDER natality database, a publicly accessible de-identified dataset. Since the data are anonymous and publicly available, the study was exempt from institutional ethics committee approval and informed consent requirements in accordance with international research regulations.

Authors' contributions: Concept – BMS, DK; Design – BMS, DK, CK; Supervision – DK; Materials – BMS, CK; Data Collection and/or Processing – BMS, CK; Analysis and/or Interpretation – BMS; Literature Review – BMS, DK, CK; Writing – BMS, DK; Critical Review – DK.

Conflict of Interest: The authors declare that there is no conflict of interest.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Use of AI for Writing Assistance: No artificial intelligence (AI)–assisted technologies were used during the write up of this manuscript.

Peer-review: Externally peer-reviewed.

References

1. Kramer MS. Determinants of low birth weight: methodological assessment and meta-analysis. Bull World Health Organ 1987;65(5):663–737.
2. Lange S, Probst C, Rehm J, Popova S. National, regional, and global prevalence of smoking during pregnancy in the general population: a systematic review and meta-analysis. Lancet Glob Health 2018;6(7):e769–e76. [CrossRef]
3. Joubert BR, Håberg SE, Bell DA, Nilsen RM, Vollset SE, Midttun Ø, et al. Maternal smoking and DNA methylation in newborns: in utero effect or epigenetic inheritance? Cancer Epidemiol Biomarkers Prev 2014;23(6):1007–17. [CrossRef]
4. Philips EM, Santos S, Trasande L, Aurrekoetxea JJ, Barros H, von Berg A, et al. Changes in parental smoking during pregnancy and risks of adverse birth outcomes and childhood overweight in Europe and North America: An individual participant data meta-analysis of 229,000 singleton births. PLoS Med 2020;17(8):e1003182. [CrossRef]
5. Bailey BA, Kopkau H, Nadolski K, Dodge P. Impact of in utero tobacco exposure on fetal growth: Amount of exposure and second trimester fetal growth measurements. Neurotoxicol Teratol 2024;102:107334. [CrossRef]
6. Blatt K, Moore E, Chen A, Van Hook J, DeFranco EA. Association of reported trimester-specific smoking cessation with fetal growth restriction. Obstet Gynecol 2015;125(6):1452–9. [CrossRef]
7. Pickett KE, Wilkinson RG, Wakschlag LS. The psychosocial context of pregnancy smoking and quitting in the Millennium Cohort Study. J Epidemiol Commun Health 2009;63(6):474–80. [CrossRef]
8. Liu B, Xu G, Sun Y, Qiu X, Ryckman KK, Yu Y, et al. Maternal cigarette smoking before and during pregnancy and the risk of preterm birth: A dose–response analysis of 25 million mother–infant pairs. PLoS Med 2020;17(8):e1003158. [CrossRef]
9. Bush P, Mayhew T, Abramovich D, Aggett P, Burke M, Page K. A quantitative study on the effects of maternal smoking on placental morphology and cadmium concentration. Placenta 2000;21(2-3):247–56. [CrossRef]
10. Polakowski LL, Akinbami LJ, Mendola P. Prenatal smoking cessation and the risk of delivering preterm and small-for-gestational-age newborns. Obstet Gynecol 2009;114(2 Part 1):318–25. [CrossRef]
11. Teasdale F, Ghislaine J-J. Morphological changes in the placentas of smoking mothers: a histomorphometric study. Neonatology 1989;55(4-5):251–9. [CrossRef]
12. Pintican D, Poienar AA, Strilciuc S, Mihu D. Effects of maternal smoking on human placental vascularization: A systematic review. Taiwan J Obstet Gynecol 2019;58(4):454–9. [CrossRef]
13. Larsen LG, Clausen HV, Jønsson L. Stereologic examination of placentas from mothers who smoke during pregnancy. Am J Obstet Gynecol 2002;186(3):531–7. [CrossRef]
14. Witt SH, Frank J, Gilles M, Lang M, Treutlein J, Streit F, et al. Impact on birth weight of maternal smoking throughout pregnancy mediated by DNA methylation. BMC Genomics 2018;19(1):290. [CrossRef]
15. Lamm SH, Ferdosi H, Boroje IJ, Afari-Dwamena NA, Qian L, Dash ED, et al. Maternal tobacco use: A third-trimester risk factor for small-for-gestational-age pregnancy outcome. Prev Med Rep 2020;18:101080. [CrossRef]
16. Yan J, Groothuis PA. Timing of prenatal smoking cessation or reduction and infant birth weight: evidence from the United

Kingdom Millennium Cohort Study. Matern Child Health J 2015;19(3):447–58. [CrossRef]

17. Kataoka MC, Carvalheira APP, Ferrari AP, Malta MB, de Barros Leite Carvalhaes MA, de Lima Parada CMG. Smoking during pregnancy and harm reduction in birth weight: a cross-sectional study. BMC Pregnancy Childbirth 2018;18(1):67. [CrossRef]
18. Haddow JE, Knight GJ, Palomaki GE, Kloza EM, Wald NJ. Cigarette consumption and serum cotinine in relation to birthweight. Br J Obstet Gynaecol 1987;94(7):678–81. [CrossRef]